Genomics Literacy Matters: Supporting the Development of Genomics Literacy Through Genetics Education Could Reduce Cognitive Forms of Racial Prejudice

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Abstract: Some genetics educators have recently argued that improving students' genomics literacy could prevent students from developing erroneous beliefs about social identity, such as the belief that racial groups differ cognitively and behaviorally because of their genes; a belief called genetic essentialism. To date, however, little research has explored if or how a conceptual understanding of genomics protects against the development of genetic essentialism. Using a randomized control trial (RCT) (N = 721, 9^{th}-12^{th} graders), we explore if students with more genomics literacy are more able to conceptually change their genetic essentialist beliefs after engaging in a learning experience designed to refute essentialist thinking. The results of the RCT demonstrated that students with higher genomics literacy (relative to those with lower genomics literacy) exhibited greater reductions in the perception of racial differences and greater reductions in belief in genetic essentialism after learning about patterns of human genetic variation. These results suggest that genetics education can protect students from developing a belief in genetic essentialism when it provides them with opportunities to learn multifactorial genetics and population thinking in conjunction with how these concepts refute essentialist thinking.

Keywords: Genetics Education, Genetic Variation, Multifactorial Genetics, Genetic Essentialism, Population Thinking

Introduction

Discrimination, violence, and genocide are regularly justified through *genetic essentialism* (Jackson Jr. & Depew, 2017). Psychologists (e.g. Dar-Nimrod & Heine, 2011) define genetic essentialism as the belief that people of the same race share some set of genes that make them physically, cognitively, and behaviorally uniform, but different from other races. Consequently, genetic essentialists believe that complex traits are influenced little by the social environment (Dar-Nimrod & Heine, 2011). Both of these beliefs make genetic essentialists prone to the *naturalistic fallacy*—that racial disparities need not be eliminated because they are an immutable result of human genetics (Lynch et al., 2018). Unsurprisingly, then, belief in genetic essentialism predicts opposition to racially ameliorative policies in white (Byrd & Ray, 2015) and non-white adults in the United States (US) (Soylu Yalcinkaya et al., 2017). This is troubling, given that estimates suggest that 20% of non-black US citizens believe in genetic essentialism (Morning et al., 2019).

Yet, 21st century genomics and mid-20th century population genetics have both revealed that genetic essentialism is genetically inaccurate (Jackson Jr. & Depew, 2017). First, it overestimates the amount of genetic differentiation between human races (Graves, 2015; Rosenberg, 2011). Second, it underestimates the amount of genetic variation among same race individuals (Graves, 2015; Rosenberg, 2011). Third, it ignores the well-established finding that complex human traits, like IQ, are malleable and responsive to social-environmental conditioning (Bratsberg & Rogeberg, 2018; Moore & Shenk, 2017; Tucker-Drob & Bates, 2016).

Very few science students appear to possess this knowledge, however. For example, Bowling et al. (2008) found that 75% of introductory biology and genetics students in college did not know that there is proportionally more genetic variation within ethnic groups than between them. Congruently, Hubbard (2017) found that 29% of anthropology students incorrectly believed

that there is proportionally more biological difference between two races than between individuals within a single race. Studies have documented similar misunderstandings of human genetic variation in US high schoolers (Author 1, 2017; Author 1 et al., 2019). A non-trivial proportion of secondary science students (i.e., likely ≥ 25%) also exhibit gene-determinist reasoning about the relationship between genes and complex human traits (see Stern & Kampourakis, 2017, p. 201). These misunderstandings, as we argue below, are the result of a deficient genetics curriculum.

Despite the social danger of genetic essentialism, its scientific flaws, and evidence that science students are unaware of its dangers and flaws (Author 1, 2015), little is known about how genetics education could be used to reduce belief in it. If students develop an understanding of the complex relationship between genetic variation and trait variation, then can this understanding reduce belief in genetic essentialism? If so, then how? In this study, we test the hypothesis that students need to develop two distinct forms of genomics literacy through genetics education to reduce their belief in genetic essentialism. On the basis of experimental results consistent with this hypothesis, we argue that supporting the development of genomics literacy through genetics education could reduce the prevalence of belief in genetic essentialism during adolescence.

What is Genomics Literacy and Why Does it Matter?

Genomics literacy is a domain specific form of scientific literacy that can be understood in terms of its fundamental and derived senses. The fundamental sense of scientific literacy is the ability to read and write in a particular scientific discipline (Norris & Phillips, 2003). Being knowledgeable in a specific domain of science is *derived* from this fundamental sense (Norris & Phillips, 2003). Derived genomics literacy therefore refers to a learner's domain specific knowledge about genomics. Fundamental genomics literacy refers to the ability to make sense of genomic information in written and oral communications. Derived and fundamental forms of

genomics literacy are interrelated because: (i) new scientific knowledge is derived from a socio-constructivist learning process involving reading, writing, and talking about scientific concepts and data (Norris & Phillips, 2003); and (ii) the ability to construct new conceptual understandings is contingent upon a learner's prior knowledge (Bransford, 2000). In this study, we focus on the derived form of genomics literacy, or a learner's domain specific knowledge about genomics[1].

Derived genomics literacy is needed to make sense of many issues about race and genetics in modern society. Scholars have demonstrated that the amount of articles discussing research on race and genomics has steadily increased in the 21st century (Phelan, Link, & Feldman, 2013). These articles describe direct-to-consumer genetic ancestry testing, racial health disparities, DNA testing in the criminal justice system, and the polygenic basis of educational attainment (Bubela & Caulfield, 2004; Martschenko et al., 2019; Phelan et al., 2014; Roth et al., 2020; Whitmarsh & Jones, 2010). Studies also demonstrate that exposure to information about race in news articles about genetics research can unintentionally increase belief in genetic essentialism through psychological priming (Lynch et al., 2008; Phelan et al., 2013). Critically, though, the effect of media on consumers' essentialist beliefs appears to be influenced by derived genomics literacy.

Take, for example, direct-to-consumer genetic ancestry testing. Nationally representative experiments suggest that when adults are exposed to information about the methods and results of genetic ancestry tests *conducted on other people*—as when the results of such tests are communicated through television commercials or shows— it exacerbates viewers' belief in genetic essentialism (Phelan et al., 2014). Yet, studies also suggest that adults who understand that genes are segments of DNA within the nuclei of cells, and who also know that human populations are genetically similar, exhibit declines in belief in essentialism after making sense of *their own genetic ancestry test results* (Roth et al., 2020). In contrast, adults lacking this knowledge show

increases in belief in genetic essentialism after making sense of such results (Roth et al., 2020). Thus, genomics literacy matters not only because it helps people make sense of ancestry testing, it also matters because it moderates how this information influences belief in genetic essentialism.

Because of such findings, legal scholars and philosophers of science contend that exposure to genomic science in the media can unintentionally increase public belief in genetic essentialism, which, in turn, can bias the public against policies designed to redress inequality (Fox, 2019; Kitcher, 2001). Studies have not yet revealed if genetics education protects the public from these effects through its impact on genomics literacy. Yet, it is hard to believe that formal genetics education is irrelevant when it comes to the problem of public belief in genetic essentialism.

Genetics Education and Genomics Literacy

We contend that the kind of derived genomics literacy that genetics education helps students to develop can either contribute to this problem[2] or to its solution. For example, Dougherty (2009), Jamieson and Radick (2013), and Stern and Kampourakis (2017) argue that the genetics curriculum may implicitly encourage students to develop essentialist beliefs because of its myopic focus on Mendelian inheritance. They contend that when students learn to view trait variation through a Mendelian lens, they begin to view human variation as discrete rather than continuous. In turn, this view reinforces the idea that there are "genes for" traits, and thus, that genes powerfully influence complex trait variation without any moderating influence of the social environment. Consequently, these scholars have proposed that school genetics should teach students concepts such as gene-environment interactions, polygenic risk, and the limits of Mendelian inheritance.

Put simply, these proposals contend that school genetics should teach students multifactorial genetics. This is the idea that multiple genetic and environmental factors influence the probability of developing certain trait expressions. Most human traits are not determined by a

single gene with two alleles (dominant and recessive) (Kampourakis, 2017). Rather most traits are polygenic, meaning they are influenced by hundreds or thousands of alleles, which, when combined, have a relatively smaller effect on trait variation (Bush & Moore, 2012) than social or environmental factors. In turn, this means that complex traits are malleable (Moore & Shenk, 2017). This is the kind of knowledge that scholars have proposed to emphasize more in the genetics curriculum. From here forward, we refer to this kind of derived literacy as *standard genomics literacy,* because of a need to emphasize it more in the standards that frame science curricula.

The majority of US genetics education standards that inform curricula do a poor job of dealing with both Mendelian and multifactorial models of inheritance (Dougherty et al., 2011; Lontok et al., 2015). In particular, Dougherty et al. (2011) show that the genetics standards in virtually every US state, including the Next Generation Science Standards (NGSS), either omit or under-emphasize concepts about genetic complexity, the importance of environment to phenotypic variation, and differential gene expression. Furthermore, a review of the last 20 years of contemporary genetics education research by Stern and Kampourakis (2017) indicates that genetics education research has not adequately addressed how to account for genetic essentialist biases in the design of genetics curricula. Nor has it extensively explored how to help students understand the complexity of multifactorial genetics through curriculum and instruction. Instead, several studies show that students are exposed to teachers who believe in genetic essentialism and to curricula that have genetic essentialist ideas embedded in them (Stern & Kampourakis, 2017).

When these factors are combined, it could mean that school genetics currently does more to promote belief in genetic essentialism than to protect against it (Author 1, 2015b). For example, randomized control trials (RCTs) carried out in schools demonstrate that exposure to certain content in the Mendelian genetics curriculum—such as racial differences in genetic disease

prevalence—can cause US middle and high school students to grow in their belief in genetic essentialism (Author 1, 2014, 2016, 2017). As for teachers, Castéra and Clément (2014) estimate that depending on the country, anywhere from 3–62% of biology teachers believe that, "Ethnic groups are genetically different and that is why some are superior to others". It is therefore unsurprising that developmental psychologists have documented a sharp increase in the tendency to explain complex human traits with genes beginning at age 11, when many US science students are first introduced to Mendelian genetics by their teachers (Meyer et al., 2020).

Altogether, this review reveals that genomics literacy matters not only because it is required to make sense of socioscientific issues related to genetics. It also matters because of its relationship to genetic essentialism—a cognitive form of prejudice. By helping students to develop an understanding of the complex relationship between genetic variation and trait variation through genetics education, it might be possible to reduce student belief in genetic essentialism. If correct, then what kind of derived genomics literacy might produce this humane effect?

Toward A More Humane Conception of Genomics Literacy

Our conception of derived genomics literacy begins by improving upon the conception advanced by Boerwinkel et al. (2017). In a Delphi study of 57 genetics educators, Boerwinkel et al. (2017) arrived at a consensus definition of derived genomics literacy for 21st century life. They claimed that a genetically informed individual should possess an understanding of multifactorial genetics. They also agreed that students needed to be prepared for discussions of genetic differences between racial groups through their genetics education. However, Boerwinkel et al. (2017) noted that a shortcoming of their consensus definition was that it was not based in a concept of evolutionary variation, such as population thinking. Nor, did Boerwinkel et al. (2017) elaborate

the anti-essentialist connections between race, population thinking, and multifactorial genetics. We now unpack these connections to contextualize our conception of *humane genomics literacy*.

Population thinking is integral to evolutionary theory because it is the idea that populations are not genetic types, rather they are aggregates of genetically varying individuals (Mayr, 1982). Ernst Mayr, who co-developed the modern evolutionary synthesis (MES), argued that population thinking was antithetical to essentialism (Mayr, 1982). Population geneticist, Theodosius Dobzhansky, who co-developed MES, further argued that genetics education needed to help the public understand population thinking because this concept could help to reduce racial prejudice (Jackson Jr. & Depew, 2017). An understanding of population thinking means that an individual knows how to conceptualize patterns of genetic variation *within* and *between* populations.

When it comes to human genetics, population thinking is concerned with the 0.1% of the human genome that differs between any two humans (Rosenberg, 2011). Genetic variation within human populations is a measurement of the amount of loci in variable DNA that differs, on average, when comparing the genomes of individuals of the same population (Rosenberg, 2011). Between group variation refers to the extra amount of loci that differ, on average, when comparing the genomes of individuals in different human populations (Rosenberg, 2011). These two forms of variation are proportionally related because the total variation in the human genome (0.1%) can be partitioned into a within-group and a between-group component (Rosenberg, 2011). On average, across all loci, 95.7% of genetic variation occurs within geographic populations of humans and an extra 4.3% of genetic variation occurs between such groups (Rosenberg et al., 2002). So, proportionally speaking, there is more genetic variation within human groups than between them. Genetic essentialism predicts the opposite pattern—more variation between than within groups—which is why population thinking refutes genetic essentialism (Lewontin, 1972).

These patterns of genetic variation, in turn, affect causal inferences about the relationship between human genetic variation and human trait variation because multifactorial genetics is a statistical science based in population thinking. To establish that alleles influence a trait, modern researchers use Genome Wide Association Studies (GWAS). These studies attempt to establish that genetic variation *within* populations correlates with trait variation after controlling for factors that vary *between* populations (i.e., population stratification) (Bush & Moore, 2012).

One important genetic factor that is controlled is linkage disequilibrium (LD), which occurs when an allele at one locus is inherited along with an allele at another locus within a population (Bush & Moore, 2012). Genetic recombination tends to break down LD within a population after many generations of reproduction (Bush & Moore, 2012). Thus, older populations, such as those within Africa, tend to have less LD than younger populations, such as those residing outside of Africa (Bush & Moore, 2012). If LD is not controlled for, then GWAS will produce spurious genetic-trait associations (i.e., an allele's effect could actually be due to a linked locus). This means that GWAS can only explain the trait variance associated with alleles *within a single population*, and only if they have controlled for genetic factors that vary *between populations*, like LD.

Yet, many populations also differ in the social-environmental conditions they experience (Rosenberg et al., 2018). Any estimate of the genetic contribution to trait variance between racially-defined populations is therefore confounded by the way in which racism and discrimination have produced different environments for people of different races (Markus & Moya, 2011). For example, studies have found that 11% of the variation in educational attainment is associated with polygenic variation within European Americans (23andMe Research Team et al., 2018). However, the same polygenic markers capture only 1.2% of the educational variation of African Americans (23andMe Research Team et al., 2018). This discrepancy has to do with

confounding created by g-e correlation (23andMe Research Team et al., 2018). Segregation and discrimination make the schooling environments of black and white Americans drastically different (e.g., see Canning et al., 2019; Darling-Hammond, 2010; Reardon et al., 2019) and this could differentially affect the magnitude of the genetic contribution to educational attainment *within* each group. Therefore, it is unclear if GWAS will ever produce an unconfounded estimate of the genetic contribution to *variation between races* because individuals inherit their genes (g) and environments (e) together, and this g-e correlation differs systematically along with allele frequencies and LD across racially-defined groups (Rosenberg et al., 2018).

For these reasons, in the 21st century, The National Human Genome Research Institute (Dressler et al., 2014) and The American Society for Human Genetics ("ASHG Denounces Attempts to Link Genetics and Racial Supremacy," 2018) still contend that population thinking and multifactorial genetics are integral to anti-racist genomics literacy (Dressler et al., 2014). These concepts serve this purpose because they show that essentialists are incorrect in believing that: (i) people of the same race are uniform; (ii) races are discrete, non-overlapping categories; (iii) genes are the single best explanation for racial disparities.

Standard Versus Humane Genomics Literacy

Knowing how population thinking and multifactorial genetics refute genetic essentialism is what we call *humane genomics literacy*. Humane genomics literacy is related to, yet distinct from, standard genomics literacy. As argued earlier, standard genomics literacy is the kind of knowledge that scholars have argued is missing or underemphasized in standards and curricula. It is a story about how trait variation is more complicated than the Mendelian explanation for it. The story is more complicated because it requires students to integrate molecular concepts, multifactorial concepts, and population thinking. Humane genomics literacy complicates this story

further, by asking the learner to explore how these concepts refute the genetic essentialist assumptions used by white supremacists. Table 1 summarizes each of these literacy conceptions.

Table 1. *Definitions of fundamental, derived, standard and humane genomics literacies*

Fundamental Literacy The ability to read and write in a particular scientific discipline, such as genomics.	
Derived Literacy Being knowledgeable in a specific domain of science, such as genomics.	
Standard Genomics Literacy A kind of derived genomics literacy that scholars have proposed emphasizing more in genetics standards and curricula, which includes ideas such as multifactorial genetics and population thinking. It is not structured for the purpose of refuting belief in genetic essentialism.	**Humane Genomics Literacy** Standard genomics literacy that is structured to refute essentialist thinking. It is the knowledge of how multifactorial genetics and population thinking refute the assumptions of genetic essentialism. This knowledge is humane because it is oriented toward reducing racism.

Table 2 describes the big ideas that constitute the derived sense of humane genomics literacy. Individuals who possess the derived form of humane genomics literacy understand *how* population thinking and multifactorial genetics refute genetic essentialism. In contrast, individuals who possess the derived form of standard genomics literacy understand population thinking and multifactorial genetics, but they *do not know how* these ideas refute genetic essentialism.

A person could therefore have a high degree of standard genomics literacy and nevertheless believe in genetic essentialism. For example, someone might have high standard genomics literacy about dog breeds and still believe in genetic essentialism (Norton et al., 2019). This could occur because patterns of genetic variation in dog breeds are the opposite of the patterns in humans (Norton et al., 2019). There is more genetic variation between dog breeds than within them (Norton et al., 2019). If a person incorrectly applied standard genomics understandings of dogs to humans, then they would believe essentialism is correct (Norton et al., 2019). But, it would be much more difficult for a person to develop humane genomics literacy and still believe in genetic essentialism because this knowledge is conditionalized on refuting racism. Humane genomics literacy is a form of standard genomics literacy that is explicitly structured for the purpose of reducing racism.

Table 2. The derived sense of humane genomics literacy.

Big Ideas	Description
Population Thinking makes it wrong to claim that people within a racial group are genetically uniform, and that racial groups are genetically discrete.	Only 0.1% of the human genome differs between any two randomly picked humans. When geneticists analyze variable DNA, they have found, repeatedly, that continental populations of humans exhibit low levels of genetic differentiation because there is proportionally more genetic variation within human populations (95.7%) than between them (4.3%) (Graves, 2015; Rosenberg et al., 2002). This replicated finding results from three important patterns in the distribution of alleles in human populations (Rosenberg, 2011). First, across loci in the human genome, populations of people tend to have the same alleles, but they differ in the proportion of individuals within each population who have certain alleles (Rosenberg, 2011). Second, private alleles that are found in only one human population are exceedingly rare (7.53% of alleles in the genome) and, on average, are only possessed by 1.65% of people in any single population (Rosenberg, 2011). Third, the amount of genetic variation within human populations declines slightly as one samples indigenous populations living further from Africa because of the combined influences of migration out of Africa, genetic drift, and the founder effect (Rosenberg, 2011). While these patterns mean that there is a population structure in humans that loosely corresponds with the US government's system of racial categorization, it also means that genetic essentialism is inaccurate. For example, it is incorrect to assume that racial stereotypes are true because individuals of the same race are genetically uniform due to the fact that most genetic variation is found among individuals of the same group. Likewise, the fact that most alleles are widely distributed across groups and that private alleles are rare indicates that racially-defined groups are alike in their variable DNA.
Multifactorial Genetics makes it difficult to explain that racial disparities are simply the result of genes.	Since complex human traits are not Mendelian it is incorrect to argue that races differ in complex and humanly important ways because of allele frequency differences at a single locus. Instead, complex traits are best explained by multifactorial models of inheritance that include large environmental effects, small genetic effects, gene-environment interactions, and many other unknown factors (Duncan & Keller, 2011). This means that complex human traits, like IQ, are malleable and their association with genetic variation is influenced by the social environment (Bratsberg & Rogeberg, 2018; Tucker-Drob & Bates, 2016). Thus, many genes do not have a stable impact on complex traits. Their impact is contingent on the environment (Moore & Shenk, 2017). It is therefore a distortion of the limits of genetic knowledge to claim that racial disparities are caused by genes when the social environments of different racial groups differ so greatly in the US (Markus & Moya, 2011). For example, many studies demonstrate that racial disparities are the result of modifiable social factors like segregation (Reardon et al., 2019) or discriminatory beliefs and attitudes (Canning et al., 2019; Markus & Moya, 2011). Furthermore, polygenic contributions to group level differences in complex traits are predicted to be small, possibly spurious, and dependent on the environment, according to population genetic theory (Rosenberg et al., 2018). Thus, while genes may have a predictable influence on trait variation among individuals of the same race who experience the same social-environment, one should be skeptical of anyone who claims that genes are the best explanation for disparities that exist between races. The differences in social environments between races that have resulted from systemic racism and discrimination in the US make it methodologically and ethically impossible to conduct a fair scientific experiment that would conclusively prove that genes are the cause of racial inequality (Author1, 2015a; Feldman & Lewontin, 1975; Goldsby, 1973; Graves, 2015; Rosenberg et al., 2018).

How These Genomics Literacies Could Affect Belief in Genetic Essentialism

We hypothesize that standard genomics literacy *enables* a reduction in essentialism. Developing it will not necessarily reduce a person's belief in genetic essentialism, but it will increase the probability that a person can change their belief in essentialism. We further

hypothesize that humane genomics literacy is more easily developed if one already has standard genomics literacy because of an expertise effect. Therefore, we predict that these two forms of genomics literacy will interact when learning experiences elicit and/or build them. This interaction should then lead to a reduction in belief in genetic essentialism because of psychological mechanisms specified by genetic essentialism theory (Dar-Nimrod & Heine, 2011).

Genetic essentialism theory (Dar-Nimrod & Heine, 2011) contends that exposure to genetic information affects belief in genetic essentialism through its impacts on how individuals perceive the relationship between genes and traits. Information that leads learners to believe that there is a *specific*, *proximate*, *stable* and *immutable* relationship between a gene and a trait tends to increase belief in genetic essentialism through a mechanism based in causal reasoning (Lynch et al., 2018). Information that leads learners to believe that individuals of the same group are genetically *uniform* and that different groups are genetically *discrete* increases belief in genetic essentialism through a mechanism based in social categorization (Lynch et al., 2018). Therefore, it may be possible to run these two mechanisms in reverse and reduce belief in genetic essentialism.

Preventing Genetic Essentialism Through Causal Reasoning and Social Categorization

Theoretically, it is probable that the development of standard genomics literacy influences causal reasoning in a way that reduces belief in essentialism. For example, developing the multifactorial understandings that (1) most traits are polygenic, (2) polygenic factors influence risk in a non-determinist way, and (3) the social/external environment interacts with genes to affect trait variation means developing a mental model of inheritance where the relationship between genes and traits is less *specific* and *proximate* than it is in Mendelian genetics. Developing this knowledge should then lead students to believe that the relationship between genes and traits is *unstable,* because the effect of genes varies across different environments. Since people perceive

environmental factors as more changeable than genes (Lynch et al., 2018), students who understand multifactorial models of inheritance should also believe that complex traits are *malleable* and not genetically *pre-determined*. Less belief in the *proximity*, *stability*, *immutability*, and *determinative* power of genes should make genes a poor explanation for social inequalities.

Developing population thinking could also affect belief in genetic essentialism by influencing social categorization. For example, developing the understanding that genetic variation exists within populations could reduce typological thinking and beliefs about the uniformity of a population. Likewise, developing the understanding that a small extra amount of genetic variation occurs between human populations could lead to a reduction in the belief that human populations are discrete, non-overlapping, categories. These effects could, in turn, reduce genetic essentialism.

Evidence supports both of these hypotheses. With regards to causal reasoning, Jamieson and Radick (2017) used a non-randomized comparative design where undergraduates (N = 56) learned genetics from a standard Mendelian curriculum or from a multifactorial curriculum emphasizing standard genomics literacy. Neither curriculum addressed population thinking or race. Students completed pre and post surveys about their endorsement of genetic essentialism. Although there was selection bias of participants into treatment conditions in the quasi-experimental design used by Jamieson and Radick (2017), students did not differ significantly in genetic essentialism before treatment. Yet, afterwards the students who received the standard multifactorial intervention had significantly lower belief in essentialism than those who received the Mendelian curriculum. This result tentatively supports the claim that belief in genetic essentialism can be prevented when causal reasoning is influenced by an understanding of multifactorial complexity, which is one component of standard genomics literacy.

A handful of studies have also explored whether a humane genetics education emphasizing

humane population thinking (Table 2) can decrease belief in genetic essentialism by affecting beliefs related to social categorization. In three different RCTs, Author 1 et al. (2019) demonstrated that teaching students about genetic variation within and between US census races can significantly reduce belief in genetic essentialism of race. In their first study, they randomized 8th and 9th grade students (N = 166) into separate classrooms to learn for an entire week either about the topics of: (i) patterns of human genetic variation; or (ii) patterns of climate variation. Both treatments used identical instructional frameworks and differed only in content objectives. In the first RCT using these two interventions, Author 1 et al. (2019) demonstrated that students who learned about human genetic variation (compared to the control) had significantly reduced scores on an instrument assessing their perception of genetic variation between racial groups and also on a measure that included items assessing belief in genetic essentialism and belief in racial stereotypes. They then replicated these findings in two more RCTs with adults (N = 176) and with biology students (N = 721, 9th-12th graders). Through a mediation analysis they also showed that learning about human genetic variation reduced belief in genetic essentialism through its impact on how students perceived genetic variation between races. Specifically, when students learned about genetic variation within and between human races, it reduced their perception of racial discreteness, which reduced their belief in genetic essentialism. These results support the claim that belief in genetic essentialism can be prevented when social categorization is influenced by an understanding of population thinking, which is one component of humane genomics literacy.

The Interaction of Standard and Humane Forms of Genomics Literacy

At present, we still do not know if or how *prior differences* in the standard genomics literacy of students interacts with curriculum and instruction emphasizing humane genomics literacy to create differential reductions in student belief in genetic essentialism. This prediction is

grounded in the literature on expertise. This extensive literature, summarized by Bransford (2000), has shown that children and adolescents can develop conditionalized knowledge and problem-solve like experts through instruction that helps them to differentiate domain-specific knowledge. For example, studies have established that domain specific prior knowledge allows one to construct more meaning from science texts (Ozuru et al., 2009; Tarchi, 2010). Studies have also found that possessing more standard genomics knowledge improves understanding of print and oral communications about genomic information (Lea et al., 2011). Relative to students with low levels of standard genomics literacy, those with high levels should be able to construct more conditionalized knowledge about how population thinking refutes genetic essentialism from a humane genetics curriculum because their prior knowledge enhances their reading comprehension.

In turn, this process should lead to a greater conceptual change in the racial thinking of students with high standard genomics literacy compared to those with low standard genomics literacy. For example, Gregoire's (2003) cognitive affective model of conceptual change predicts that the impact of an educational reform message on the conceptual change process depends on whether learners respond to it with a threat or challenge appraisal. Students who have insufficient prior knowledge to understand such ideas may perceive the learning as a threat to their ego. A threat appraisal could lead these students to heuristically process the information, which leads to no belief change or superficial change (i.e., assimilation). In a challenge appraisal, however, students are more likely to systematically process the information presented to them because they possess the prior knowledge necessary for learning. Systematic processing will, in turn, increase the probability that students revise their beliefs (i.e., accommodation).

A humane genetics education could threaten students because it requires them to interrogate genetic beliefs about racial identity, especially those that have been used to buttress the

self-esteem and privilege of white people by derogating the status of people of color. If students do not possess the prior knowledge for understanding the content of a humane genetics education, then they might feel incompetent while learning, adding more ego-threat to an already threatening learning experience, and this extra threat could altogether prevent a change in essentialism. Standard genomics literacy may therefore be necessary, but not sufficient, for the conceptual change of genetic essentialism through a humane genetics education. It matters not only because of an expertise effect that influences the development of humane genomics literacy, but also because this expertise buffers against any additional ego threat that may be created as one makes sense of the complex conceptual terrain in a humane genomics education.

Research Questions

We ask two research questions (RQ) generated by our hypothesis in the present study. After learning how patterns of human genetic variation refute essentialism, are students with high standard genomics literacy (SGL) (relative to students with low SGL):

(RQ1) more likely to change their perception of genetic variation between races?

(RQ2) more likely to change their belief in genetic essentialism?

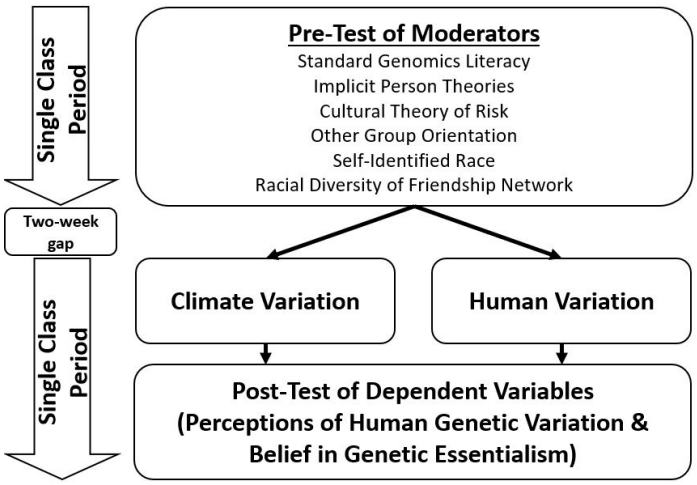
At this point, one might argue that the questions generated by our hypothesis are circular in nature. For example, one might claim that anyone with high SGL would be less likely to believe in any genetic fallacy. Or, they might argue that if genetics education improves humane genomics literacy, then this improvement will, by definition, create less belief in genetic essentialism. Yet, studies suggest that such arguments are flawed. First, in a sample of 427 Brazilian undergraduates, Gericke et al. (2017) found that SGL was not correlated with belief in genetic determination. Thus, knowing some things about genomics does not necessarily mean that one will disbelieve any genetics fallacy. Second, genomically literate people are still human, which means the reasoning

connecting their racial beliefs to their genomics knowledge will often be socially-motivated, context specific, and contingent on their identity (Keller, 2005; Morton et al., 2009). Third, when an intervention uses scientific facts to discredit a myth, it can create a novel link between the myth and the fact, which can lead the intervention to backfire through a recall error where the learner believes the myth is the scientific fact (Lewandowsky et al., 2012). Backfiring can also occur when science interventions unintentionally threaten the worldviews or the core identities of a learner (Darner, 2019). When these threats are combined with instruction that thwarts a learner's psychological needs and elicits their negative emotions, it can inhibit conceptual change (Darner, 2019). Consequently, attempting to reduce belief in genetic essentialism by increasing humane genomics literacy could backfire. Clearly then, standard genomics literacy, humane genomics literacy, and their relationship to essentialism are worthy of further research.

Methods

To answer RQs 1 and 2, we explore how a computerized intervention about human genetic variation and race interacts with the standard genomics literacy of students to affect their racial perceptions and beliefs (Figure 1). We use a special kind of RCT called an individually randomized trial with clustering (IRTC) (Kahan & Morris, 2013). In an IRTC, individuals are randomized to experimental arms within study sites and then receive a treatment (Kahan & Morris, 2013). The IRTC data that we analyze in this study was produced in the third RCT of Author 1, et al. (2019). We report new findings from this study. In the supplemental, we also corroborate these findings with new evidence from cognitive think-alouds with the intervention materials.

Figure 1. *Summary of within classroom and within racial group experimental design*



Sample Descriptions

Given that our study is attempting to establish preliminary proof of concept for our interaction hypothesis, many factors influenced our sampling decisions. White individuals in high socioeconomic status (SES) communities tend to justify racial discrimination through cognitive forms of prejudice like essentialism (Morton et al., 2009; Tawa, 2016). Furthermore, many white science teachers come from high SES communities and many of them may also hold implicit biases about the abilities of non-white students (Bryan & Atwater, 2002). The same can be said about scientists, who tend to grow up in racially and economically privileged groups (Aikenhead, 1996; Bryan & Atwater, 2002; Taie & Goldring, 2018). Scientists and science students who believe that science ability is inherited and immutable are also known to create a threatening learning atmosphere that can negatively influence the motivation and achievement of black and Latinx students (Canning et al., 2019; Leslie et al., 2015; Storage et al., 2016). We sampled students from majority-white, but racially-diverse, high SES schools because protecting students in these schools from believing in genetic essentialism is socially consequential.

The IRTC sample was a group of 9th–12th graders (n= 721) recruited from five schools in the spring of 2018. Two public high schools were sampled from major cities in Colorado (33.34%

of the sample). In California, one public high school was sampled in the San Francisco Bay Area (41.19% of the sample). We also sampled one public high school in the Boston, Massachusetts area (20.39% of the sample) and one private high school in the Washington, D.C. area (4.99% of the sample). Free and reduced-price lunch (FRL) was high at one of the Colorado sites (FRL = 66%) and low at the other (FRL = 12.1%). FRL for the remaining schools was low (Washington DC = 0%, Boston = 25.9%, and California = 6.8%). The percent of White students at each school ranged from 71% to 20%. Students self-identified their race as White (61.7%), Asian (19.8%), Mixed-Race (9.9%), Hispanic (4.9%), Black (2.4%), Pacific-Islander (0.55%), or American Indian (0.4%). The mean age of students was 15 (SD = 1.02, range 14–18) and 47.7% identified as female. Students were in 9th grade (54.1%), 10th grade (19.6%), 11th grade (25.9%), or 12th grade (0.4%).

The IRTC Design

Treatment. The human genetic variation and race treatment used in the IRTC consisted of a computerized intervention that engaged students in sense making about patterns of genetic variation within and between the US census categories. More specifically, it helped students to make sense of the big ideas on "population thinking" from Table 2 using the "time for telling" instructional framework (Schwartz & Bransford, 1998). From here forward, we refer to this intervention as the humane genomics intervention. This intervention first elicited students' essentialist beliefs about race with a discrepant event. Then, contrasting cases of genetic data (Schwartz & Bransford, 1998) were used to scaffold sense making about patterns of genetic variation within and between ancestry groups. This curricular framework was chosen to scaffold data interpretation in order to decrease the likelihood that students misconstrue data to fit their preconceptions (Chinn & Malhotra, 2002). After, students were asked to craft an argument about whether the real evidence supported or rejected claims about the uniformity of same race people

and the discreteness of different races. Following this segment, students then used the evidence they had learned to critique an essentialist argument made by a hypothetical character. Afterwards, students read a short text describing why people tend to misrepresent genetic differences within and between races when claiming that race is biologically real. For more detail on the learning theories and curricular framework of this intervention refer to Author 1 et al. (2019).

Control. We compared the learning outcomes from the humane genomics intervention to a control condition where students learned about the patterns of climate variation that support human-induced climate change. The climate variation control condition taught students about temperature and precipitation variability within and between climate zones. It additionally taught students how people tend to misrepresent those differences when claiming that climate change is not real. It also was designed with the "time for telling framework" and was therefore instructionally identical to the humane genomics intervention. Both curricular interventions were written at the 8th grade level and each took students, on average, 45 minutes to complete.

We used a climate curriculum for the comparison because it would control for ideologically motivated reasoning and the cognitive difficulty of reasoning about variation. Biological beliefs about race are ideological issues (Lewontin, 1996), and since reasoning about biological variation is cognitively complex for students (Emmons & Kelemen, 2015), a suitable control condition would need to control for the ideological and cognitive complexity of learning about genetic variation and race. For example, if ideologically motivated reasoning is triggered when students learn about race and genetics, then this could introduce heterogeneity into our data that could bias a treatment effect positively or negatively. Using a curriculum about the human causes of climate variation as a control could balance out this potential biasing effect, as climate change is known to trigger ideologically motivated reasoning in the US. (Kahan, 2016). Also, the climate variation

control has the added benefit of requiring students to make sense of temperature and precipitation variation within and between geographic areas and time periods. Thus, it also controls for the cognitive difficulty of learning about variability. Without controlling for such cognitive difficulty, variation in the cognitive load of the interventions could bias treatment effect estimates.

It might be argued that a more clinically relevant control condition would involve business-as-usual (BAU) materials involving race and genetics, such as learning about the prevalence of genetic diseases in various racial groups. However, previous studies have found that exposure to materials describing this content causes an increase in belief in genetic essentialism (Author 1, 2014, 2016, 2017). Indeed, even exposing individuals to genetic information that includes "gene for" language or a blueprint metaphor for DNA can prime belief in genetic essentialism (Lynch et al., 2008; Parrott & Smith, 2014). If these priming effects occurred in response to our control curriculum, then this effect could create a difference between conditions that is unrelated to the humane genomics intervention that used population thinking to refute essentialism. As we wanted to have an inert control, we did not use either of these BAU controls. This methodological choice further reduces the chance that our treatment effect estimates are upwardly biased. Moreover, studies have found that experiments which compare a novel science education intervention to a different intervention yield smaller treatment effects, on average, than when a novel intervention is compared to a business-as-usual (BAU) curriculum (Kowalski et al., in review). Thus, the treatment effects that we observe in this study are conservative and likely smaller than what will be observed in replications using BAU materials for comparison.

Randomization. Within each classroom individual students were randomized to either the climate curriculum (control) or the humane genetics curriculum (treatment). The entire experiment was delivered to students on tablet devices or laptops through the Qualtrics survey platform. We

used survey logic to randomize students within each classroom to experimental arms within each self-identified race (see Figure 1 for summary of design).

Dependent Variables. The items for each instrument can be found in the supplemental. Perceptions of human genetic variation (PHGV) were measured with the perceptions of biological variation measure (Cronbach's $\alpha = 0.86$) which was developed and validated to measure adolescent perceptions of genetic variation within and between races (Author 1, 2017). As in previous studies (Author 1, 2017; Author 1, et al., 2019), we averaged the between race and within race questions separately. Then, we divided the average of the between group items per student by the sum of the averages of the within and between group items per student, which yielded a single proportion for each student that could take on any value between 0 and 1. Higher scores on this instrument (0-100%) indicate that a student perceives a greater proportion of genetic variation between races relative to the total variation perceived within and between races. A score of 4.3% represents the scientifically estimated proportion of genetic variation that occurs between the continental groups commonly associated with US census races (Rosenberg et al., 2002)

Belief in genetic essentialism was measured with twelve items from two different genetic essentialism of race instruments (see Parrott et al., 2005; Williams & Eberhardt, 2008) assessing the biobehavioral and biosomatic components of this construct, which are conceptually distinct, yet related, forms of genetic essentialism (Andreychik & Gill, 2014). The correlation between these two instruments in the present study was 0.49, which supports the relatedness of these measures. Below we also show that the two instruments are statistically distinguishable. Items in this composite instrument ($\alpha = 0.84$) assessed agreement with statements such as: "two Black people will always look more similar to each other than a Black person and a White person ever

would"; "Racial differences in academic ability are caused by genetics". Items were anchored on a scale of 1 (strongly disagree) to 7 (strongly agree). Higher scores equal greater essentialism.

Standard Genomics Literacy (SGL). Population thinking was assessed through 12 multiple choice items assessing high school student's ability to reason with data about variation within and between populations of bees and flowers. Although these items are still unpublished, we refer to them as the Quantitative Reasoning in Biology Instrument, or QRB. The QRB assesses quantitative reasoning about time-series variation, co-variation, and within and between group variation in different biological systems (NSF-DRK-12 Award # 1503005). We used a subset of the items on between and within group variation in populations of bees because they assessed population thinking but they did not assess how this knowledge refuted genetic essentialism. We assessed knowledge of multifactorial genetics through a subset of 16 multiple choice items in the Genetics Literacy Assessment Instrument, or GLAI (Bowling et al., 2008), which assessed knowledge of molecular genetics, polygenic risk, and multifactorial models of inheritance. Likewise, these items did not assess how multifactorial knowledge refuted genetic essentialism. The questions and answers for each assessment item can be found in the supplemental.

To estimate trait variability in genomics literacy we performed Rasch analyses (Bond & Fox, 2015) on the GLAI and QRB items. Since we combined these items to define genomics literacy, we first conducted a Principal Components Analysis (PCA) using a polychoric correlation matrix to test for unidimensionality. The first principal component in the data explained 59% of the variance (Eigenvalue = 6.46) and a parallel analysis indicated that there was only one component in the data. Since the unidimensionality assumption was met, we then proceeded to create the genomics literacy variable using Rasch modeling (Table 3).

Table 3 summarizes the psychometric results from these analyses and the proportion of

students that answered each item correctly. A score above zero on the SGL measure means that a student has a >50% chance of correctly answering the mean difficulty item in the SGL assessment. A score <0 means that a student has a <50% chance of correctly answering the mean difficulty SGL item. The bolded items in Table 3 are those that were more difficult than the mean item difficulty from the Rasch Model. Table 3 also maps each item to the concept it assessed as well as its hypothesized relationship to an essentialist belief. Refer to the supplemental for the Wright Map, a full report of item fit statistics, and a complete list of the assessment questions and answers.

Table 3. *Mapping of standard genomics literacy items to multifactorial concepts, essentialist beliefs, and item difficulty*

Item	*p(Correct)*	*Genomic Concept Assessed*	*Related Essentialist Belief*
GLAI19	.312	Gene regulation	Stability
QRB8	.341	Between & Within Group Variation	Discreteness, Uniformity
GLAI24	.358	Between &Within Group Variation	Discreteness, Uniformity
GLAI25	.370	Molecular Genetics	Proximity
GLAI22	.392	Molecular Genetics	Proximity
QRB28	.395	Knowledge of Epistemology	N/A
GLAI23	.398	Multifactorial Model	Stability, Proximity, Immutability
GLAI21	.423	Multifactorial Model	Immutability
GLAI18	.461	Multifactorial Model	Stability, Proximity, Immutability
QRB27	.473	Knowledge of Epistemology	N/A
GLAI11	.539	Gene Therapy Technology	Pre-determination, Proximity
GLAI15	.546	Within group Variation	Uniformity
QRB12	.547	Between group variation	Discreteness
QRB9	.559	Between & Within Group Variation	Discreteness, Uniformity
QRB20	.589	Between &Within Group Variation	Discreteness, Uniformity
GLAI20	.614	Genetic Risk	Pre-determination, Proximity
GLAI28	.615	Mendelian Genetics	Pre-determination
GLAI3	.651	Polygenic Inheritance	Proximity
QRB26	.683	Between &Within group Variation	Discreteness, Uniformity
QRB15	.683	Between Group Variation	Discreteness
QRB19	.736	Between &Within group Variation	Discreteness, Uniformity
QRB25	.759	Between Group Variation	Discreteness
QRB14	.763	Within group Variation	Uniformity
GLAI10	.771	Polygenic risk	Pre-determination
QRB16	.799	Between Group Variation	Discreteness
GLAI30	.815	Molecular Genetics	N/A
GLAI1	.870	Molecular Genetics	N/A
GLAI12	.909	Genetic Risk	Pre-determination

Person Reliability	*Person Separation*	*Cronbach's Alpha*	*Mean Person Measure (logit)*	*Outfit Mean-Square Fit*
.78	1.86	.81	.51	.73 – 1.26

Looking across items in Table 3 it is apparent that questions assessing knowledge of population thinking (QRB items assessing Between & Within Group Variation) were spread evenly throughout the assessment at all levels of item difficulty. Questions assessing knowledge of molecular genetics were also spread across the item difficulty distribution, being both very difficult (GLAI 22 & 25) and very easy to answer correctly (GLAI 1 & 30). Questions about genetic risk fell below the mean item difficulty of the assessment. In contrast, questions assessing knowledge of multifactorial models of inheritance fell above the mean item difficulty. These patterns suggest that a higher SGL score means that a student is more likely to know about multifactorial models of inheritance, genetic risk, molecular genetics, and population thinking.

Statistical Independence of Instruments. By the nature of the questions asked, the genomics literacy assessment was designed to be structurally independent of the genetic essentialism scale, such that it would be possible to score high on one measure without scoring high on the other. To check whether this independence bore out, we conducted a principal components analysis augmented with a parallel analysis using all of the items composing the SGL assessment, the genetic essentialism scale, and the PHGV scale. The parallel analysis revealed that a statistically significant five-factor solution existed in the data. After rotating these factors orthogonally, we found that the within group and between group items from the PHGV loaded heavily onto the first and fourth factors, respectively, thus supporting their statistical independence. All of the SGL items loaded heavily onto factor two. The genetic essentialism items from the GBRI and RCS loaded heavily onto factor three and five, respectively, thus supporting their ability to measure the two distinct, yet related, components of genetic essentialism (i.e., biosomatic and biobehavioral essentialism). This evidence clearly undermines the claim that these variables are measuring the same underlying latent construct. This independence can further be

seen when counting and categorizing students. For example, in the total sample, 19.8% of students (n = 145) disagreed with genetic essentialism and were below average on the SGL; 9% (n = 80) agreed with genetic essentialism and were below average on the SGL; 8% (n = 58) agreed with genetic essentialism and were above average on the SGL; 58.3% (n = 437) disagreed with genetic essentialism and were above average on the SGL; and one student neither agreed or disagreed with essentialism and had average SGL. These results also demonstrate that it is not always true that someone higher in SGL will be less likely to believe in a genetic fallacy.

Covariates. To assess baseline equivalence and evaluate whether the relationship between SGL and belief in genetic essentialism was robust to confounding, we measured students on covariates at baseline, such as: (i) the racial diversity of students' friendship networks (for method see Tawa, 2016); (ii) implicit person theories (IPT) of intelligence (Blackwell et al., 2007); (iii) IPT of science ability (Chen & Pajares, 2010); (iv) IPT of group behavior (Halperin et al., 2011); (v) cultural theory of risk (CTR) (Kahan et al., 2007); (vi) interest in interracial socialization measured via the other group orientation scale (OGOS) (Phinney, 1992); and (vii) self-identified race. Covariates i-vii are associated with belief in genetic essentialism. Measurement instrument items can be found in the supplemental, along with theoretical and empirical justifications for how these constructs are related to belief in genetic essentialism.

Baseline Equivalence. A Multivariate Analysis of Variance demonstrated that the two experimental arms in the IRTC did not differ significantly in IPT, CTR, OGOS, SGL, or racial diversity of friendship networks prior to the start of the experiment (Pillai's trace = 0.0088, p = 0.2776). The block randomization of our design ensures that within each class there is an equal balance of students of different races in each experimental arm. Consequently, the experimental groups are baseline equivalent on all covariates assessed before the beginning of the experiment.

Statistical Analyses

To answer our questions we use generalized estimating equations (GEEs) (Zeger et al., 1988). GEEs are an extension of the generalized linear model that are used when observations are structured, or nested, within groups or individuals. We use GEEs with robust standard errors for two reasons. First, the SGL of individual students within each classroom is correlated because they learned genetics together before these studies. Failing to account for these correlations would produce downwardly biased standard errors and could lead to false conclusions about statistical significance. We avoid this problem through our use of GEEs.

In Table 4, we report results from GEEs that employed an identity link to a Gaussian distribution and an exchangeable correlation matrix to account for the clustering of students within classrooms. First, we regressed z-scores of each dependent variable (PHGV or Genetic Essentialism) onto the SGL person measure, the treatment indicator for the human genetic variation intervention (TRT, 1 = Race, 0 = Climate), and a covariate that accounted for the within classroom correlation (CORR) using equation 1, below:

$$\text{EQ1: Variable} = \beta_0 + \beta_1(\text{SGL}) + \beta_2(\text{TRT}) + \text{CORR} + \text{Error}$$

Second, we regressed each z-scored dependent variable onto SGL scores, TRT, and the interaction of the two (TRTxSGL) using equation 2, below:

$$\text{EQ2: Variable} = \beta_0 + \beta_1(\text{SGL}) + \beta_2(\text{TRT}) + \beta_3(\text{TRTxSGL}) + \text{CORR} + \text{Error}$$

To further test if the results from equation 2 were robust to possible confounding, we then added all of the covariates described earlier, as well as covariate-treatment interactions, that could confound the TRTxSGL effect (i.e. $\beta_{16\text{-}27}$) using equation 3 below:

$$\text{EQ3: Variable} = \beta_0 + \beta_1(\text{SGL}) + \beta_2(\text{TRT}) + \beta_3(\text{TRTxGL}) + \beta_4(\text{IPT}) + \beta_5(\text{CTR}) + \beta_6(\text{OGO}) + \beta_7(\text{FRIEND}) + \beta_8(\text{WHITE}) + \beta_9(\text{ASIAN}) + \beta_{10}(\text{HISPANIC}) + \beta_{11}(\text{MIXEDRACE}) + \beta_{12\text{-}15}(\text{SCHOOL}) + \beta_{16}(\text{TRTxIPT}) + \beta_{17}(\text{TRTxCTR}) + \beta_{18}(\text{TRTxOGO}) + \beta_{19}(\text{TRTxFRIEND}) + \beta_{20}(\text{TRTxWHITE}) + \beta_{21}(\text{TRTxASIAN}) + \beta_{22}(\text{TRTxHISPANIC}) + \beta_{23}(\text{TRTxMIXEDRACE}) +$$

$$\beta_{24}(\text{TRTxSCHOOL1}) + \beta_{25}(\text{TRTxSCHOOL2}) + \beta_{26}(\text{TRTxSCHOOL3}) + \beta_{27}(\text{TRTxSCHOOL4}) + \text{CORR} + \text{Error}$$

Models 1-3 in Table 4 use EQs 1-3, respectively, to model perceptions of human genetic variation (PHGV). Models 4-6 in Table 4 use EQs 1-3, respectively, to model genetic essentialism. RQs 1 and 2 are answered through the coefficient on TRTxSGL derived using EQ2. The coefficient on TRTxSGL in EQ3 allows us to check if the results used to answer RQ1 and 2 are robust to possible confounding. Additional analyses are reported in the supplemental.

Results

Compared to students who learned from the climate intervention, students who learned from the humane genomics intervention had significantly lower perceptions of human genetic variation (PHGV) scores (Cohen's d = -0.78, R^2 = 0.13) and significantly lower genetic essentialism scores (Cohen's d = -0.39, R^2 = 0.04) ($ps < 0.001$, Table 4, Models 1 and 4) after controlling for the SGL of each student. These findings are in line with our hypothesis that humane genomics literacy matters for reducing misperceptions of human genetic variation and reducing belief in genetic essentialism. In the present study, we wanted to know if these reductions were significantly greater when comparing students with higher SGL to those with lower SGL.

To initially test these predictions, we estimated the correlation between SGL and belief in genetic essentialism or PHGV within each experimental condition. In the control condition, the correlations between SGL and PHGV (β = -0.031, R^2 = 0.017, p = 0.019), and between SGL and genetic essentialism (β = -0.136, R^2 = 0.025, p = 0.013), were relatively weak, but statistically significant and in the predicted negative direction. In the treatment condition, the correlations between SGL and PHGV (β = -0.088, R^2 = 0.13, $p < 0.001$), and between SGL and genetic essentialism (β = -0.447, R^2 = 0.24, $p < 0.001$), were also statistically significant, and in the predicted negative directions. These correlations were approximately 3 times more negative than

they were in the control condition, and also much stronger, in terms of variance explained. If the correlation between SGL and the dependent variables differed by experimental condition (with the treatment slope being more negative than the control slope), then this would provide statistical evidence in support of our general hypothesis.

Table 4. *Output of GEE Models Testing Research Questions 1 and 2*.

	Perceptions of Human Genetic Variation (PHGV)			Belief in Genetic Essentialism		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	β/(se)	*β*/(se)	*β*/(se)	*β*/(se)	*β*/(se)	*β*/(se)
SGL	-0.224***	-0.120*	-0.057	-0.288***	-0.123*	0.024
	(0.029)	(0.047)	(0.044)	(0.036)	(0.053)	(0.049)
TRT	-0.732***	-0.630***	-0.567	-0.378***	-0.216**	-0.585
	(0.075)	(0.077)	(0.860)	(0.068)	(0.079)	(0.854)
TRTxSGL		-0.203**	-0.205**		-0.319***	-0.340***
		(0.070)	(0.073)		(0.074)	(0.074)
N	721	721	721	721	721	721

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Note: We suppress the output for the main effects of covariates and each of their interactions with the treatment in Models 3 and 6.

Research Question One

The output of model 2, in Table 4, explores if these correlations differed for PHGV (see also Figure 2a). As predicted, the results of model 2 demonstrate that the humane genomics intervention caused significantly greater reductions in PHGV comparing students high in SGL to students low in SGL because of the negative TRTxSGL interaction effect (β = -0.20, SE = 0.07, z = -2.91, p = 0.004, 95% CI [-.341, -.066], R^2 = 0.01).

Research Question Two

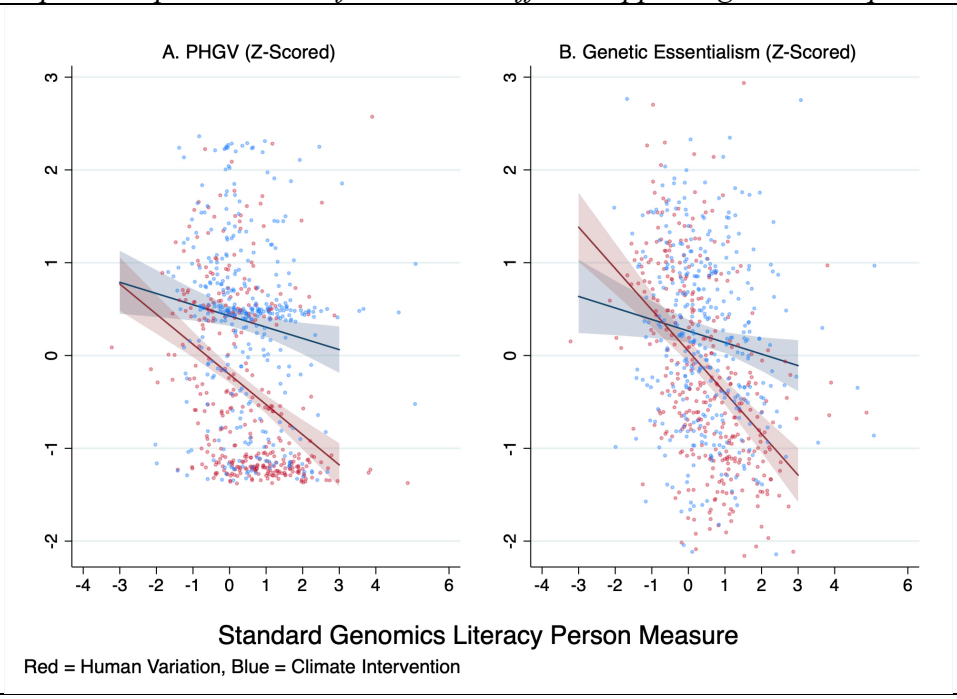
The output of model 5, in Table 4, explores if these correlations differed for genetic essentialism (see also Figure 2b). As predicted, the results of model 5 demonstrate that the humane genomics intervention caused significantly greater reductions in genetic essentialism comparing students high in SGL to students low in SGL because of the negative TRTxSGL interaction effect (β = -0.32, SE = 0.074, z = -4.30, $p < 0.001$, 95% CI [-.465, -.173], R^2 = 0.03). Among students who had less than a 50% chance of correctly answering the mean difficulty SGL item, there was

no effect of experimental condition (β = .09, SE = 0.127, z = 0.73, p = 0.468).

Robustness of Effects

The significant negative interaction between SGL and the humane genomics intervention (TRTxSGL) on each variable also persisted in a model that controlled for the main effects of possible confounding covariates and their respective interactions with treatment as well as the main effects of school sites and their interactions with treatment (Table 4, Models 3 & 6). Furthermore, all of the TRTxRace interaction terms in EQ3 were non-significant (i.e. $\beta_{8\text{-}11}$), meaning that the main effect of treatment did not depend on a student's self-identified race. Even when we added additional 3-way interactions between student racial identities, SGL and treatment into EQ3, they, too, were insignificant (e.g., all TRTxSGLxRace interaction effects: $0.14 < p < 0.95$). Likewise, adding additional interaction terms between covariates, school sites, and SGL into EQ3 (e.g., GLxIPT or GLxSchool) does not change the direction or magnitude of the TRTxSGL interaction effects. Thus, the TRTxGL effects persist in all of our tests for confounding.

Figure 2. *Graphical representation of interaction effects supporting research questions 1 and 2*



Altogether, these findings support our hypothesis because students with higher SGL (relative to those with less) were significantly more likely to change their perception of human genetic variation (RQ1) and their belief in genetic essentialism (RQ2) after they learned how population thinking refutes genetic essentialism. In the supplemental, we further corroborate these results with findings from cognitive think-alouds with our intervention. There, we show that high SGL students are more likely than low SGL students to actively read the humane genomics instructional materials, and are also more likely to discuss their understanding that racial groups are genetically alike while reasoning with the instructional materials.

The Number Needed to Treat (NNT)

To conceptualize the clinical significance of these findings for reducing belief in genetic essentialism, we estimated the number needed to treat (NNT). The NNT estimates how many individuals would need to be treated with an intervention in order to prevent one additional individual from developing an outcome. The outcome for this analysis was the proportion of students in the treatment (P_T) and control (P_C) who agreed with genetic essentialism. To calculate this proportion, we categorized students with average scores greater than the scale midpoint on the genetic essentialism instrument as individuals who agreed with genetic essentialism. Those with scores below the midpoint were counted as individuals who disagreed (i.e. a score < 4 = disagree, on average). We then modeled the unadjusted treatment effects with this data using a GEE with a logit link to a binomial distribution. We did this for all students in the sample, for only those students who had high SGL (person measure > 0), and for only those students who had low SGL (person measure < 0). We then used the odds ratios ($OR = (P_T/(1-P_T)) \div (P_C/(1-P_C))$) from these models to calculate the risk ratios ($RR = OR/((1-P_C) + (P_C \times OR))$) and absolute risk reductions ($ARR = P_C - P_T$) for each group of students. The RR estimates the relative increase or decrease in

the prevalence of an outcome comparing treatment and control students. The ARR estimates the raw reduction in the prevalence of an outcome caused by a treatment. The NNT is calculated using the ARR (NNT = 1/ARR). Refer to Sainani (2011; 2012) for further explanation of these equations.

For low SGL students, the humane genomics intervention produced an 8% reduction in the relative risk of agreeing with genetic essentialism, but it was not statistically significant (OR = 0.885, z = -0.43, p = 0.667, RR = 0.92, ARR = 2.5%). The NNT for this group was 40. For high SGL students, the humane genomics intervention produced a 54% reduction in the relative risk of agreement with genetic essentialism that was statistically significant (OR = 0.418, z = -2.79, p = 0.005, RR = .46, ARR = 8.2%). The NNT for this group was 12. Computing these statistics for the entire sample without considering the SGL of students resulted in a 29% reduction in the relative risk of agreement with genetic essentialism that was statistically significant (OR = 0.66, z = -2.53, p = 0.012, RR = .71, ARR = 6.1%). Disregarding SGL, the NNT for the entire sample was 16.

What do these number mean? First, let's begin with the NNT that ignores the SGL level of students (NNT = 16). This means that for every seventeen students who learned from the intervention in our sample one student was prevented from developing a belief in genetic essentialism. Comparatively, it takes forty-one low SGL students treated with the humane genomics intervention to prevent one of them from developing a belief in genetic essentialism. However, only thirteen high SGL students need to be treated with the intervention to prevent one of them from developing genetic essentialism. In short, the humane genomics intervention works 3.3 times better for high SGL students than it does for low SGL students.

Discussion

Altogether, these results suggest that standard and humane genomics literacies interact to prevent belief in genetic essentialism. We hypothesized that students higher in SGL (HISGL)

would be more capable of developing humane genomics literacy than low SGL (LOSGL) students because their greater expertise would create more reading comprehension and less ego-threat while they learned how population thinking refutes essentialism during the intervention. Thus, we predicted that HISGL students would show relatively greater reductions in their perceptions of genetic variation (PHGV) between races after experiencing the intervention. The results for RQ1 supported this prediction. Since previous studies (Author 1 et al., 2019) and genetic essentialism theory (Dar-Nimrod & Heine, 2011) have established that perceptions of the discreteness of racial categories mediate the relationship between genetic information and belief in genetic essentialism, we further predicted that HISGL students would exhibit greater reductions in belief in genetic essentialism than LOSGL students if the humane genomics intervention caused greater reductions in PHGV among them. The results for RQ2 also supported this hypothesis. In fact, we found that the humane genomics intervention had no effect on genetic essentialism among LOSGL students.

We contend that these findings are due to the relatively greater reading comprehension that HISGL students possess. Reading comprehension involves constructing a coherent mental representation that captures the intended meaning of a text (Norris & Phillips, 2003). We would expect HISGL students to have better reading comprehension than LOSGL students because prior knowledge influences a reader's ability to bridge information from different parts of a text (Ozuru et al., 2009) and to make inferences about text meaning (Tarchi, 2010). In other words, prior knowledge facilitates active reading of textual information[3]. More active reading could, in turn, mean that HISGL students took more advantage of the opportunities afforded by the curriculum to learn about genetic variation in the intended manner[3]. This would make them more likely to develop the humane form of population thinking (Table 2), and lead to a reduction in belief in genetic essentialism by triggering the mechanisms specified by genetic essentialism theory.

Better reading comprehension could also protect HISGL students from experiencing ego-threat that shuts down conceptual change. Under Gregoire's (2003) cognitive affective model of conceptual change, belief accommodation depends on whether learners respond to new information with a threat appraisal or a challenge appraisal. Threat appraisals do not lead to conceptual change. Challenge appraisals, on the other hand, increase the probability of conceptual change because they lead a learner to systematically process new information presented to them. Systematic processing occurs when students have the prior knowledge and the motivation to understand new information. The information in a humane genomics education could threaten students because it requires them to interrogate beliefs that have been used to buttress their self-esteem and/or racial privilege. The reduction in belief in genetic essentialism that was observed in HISGL students but not LOSGL students could therefore reflect how these groups appraised the information in the humane genomics intervention (i.e., threat vs. challenge). From this point of view, SGL matters for changing student belief in genetic essentialism because it confers the expertise needed to comprehend the information in a humane genomics education, which provides more protection from any ego threat that could prevent conceptual change or create backfiring.

Limitations

From a conceptual change perspective, this chain of events makes students with greater SGL *and* greater reading ability in the domain of genomics (i.e., more fundamental genomics literacy) more capable of changing their genetic essentialist beliefs through a humane genomics education[3]. To our knowledge, no scholars have defined the fundamental form of SGL. Since we did not measure this construct, it is possible that the effects we are attributing to derived SGL are driven instead by individual differences in fundamental SGL.

Relatedly, our study cannot tell us which pieces of conceptual knowledge constituting

derived SGL enabled a belief reduction in essentialism. For instance, our estimate of each student's SGL was derived from an assessment that included items on population thinking, multifactorial concepts, and molecular concepts. Future research should explore which of these domains of SGL are necessary and/or sufficient for catalyzing a reduction in genetic essentialism when they are combined with humane genomics instruction. Recent research with adults by Roth et al. (2020) suggests that molecular knowledge and population thinking are both important toward this end.

A more striking question that remains unanswered is whether or not the development of SGL alone has any *direct* relationship to genetic essentialism? As argued earlier, a theoretical basis for this relationship exists and the correlation between SGL and belief in genetic essentialism in our study was statistically significant in the control condition, but it was very weak. Since correlation does not imply causation, our study could not estimate whether SGL played a direct causal role in reducing belief in genetic essentialism. Instead, we found that greater SGL enabled greater reductions in essentialism by catalyzing the development of humane genomics literacy[3]. So, the effect of SGL on belief in genetic essentialism was indirect. Further research into these issues could inform the development of a learning progression for a humane genomics education.

Likewise, future studies need to test the domain specificity of the humane genomics literacy effects further. Our study established that the population thinking component of humane genomics literacy matters for reducing belief in essentialism, but our study did not allow us to estimate whether the multifactorial component matters as well. A 2x2 factorial design that randomizes students to a multifactorial genetics intervention (Factor 1) and/or to a population thinking intervention (Factor 2) could directly test if gaining these two components of humane genomics literacy creates larger reductions in genetic essentialism than learning about only one of these components, or none. If the present results were replicated through this study after

controlling for academic achievement in other subjects, reading ability, fundamental genomics literacy, or even IQ, then these results would strongly support our hypothesis.

At this point, one might claim that the domain-specific effects we have reported are spurious because academic performance is correlated across subject areas and it varies geographically due to district level differences in socioeconomic status (Reardon, 2016) and racial segregation (Reardon et al., 2019). Thus, it is possible that the interaction effect between SGL and the humane genomics intervention that we estimated is just a product of school level differences in academic achievement or reading ability. However, the TRTxSGL effects persisted even when we controlled for the main effects of school sites, self-identified race, and the interaction of each of these variables with experimental condition. These interactions also persisted after controlling for possible confounding covariates that differ between students, such as the main effects of implicit person theories, racial diversity of friendship networks, attitudes toward socializing across racial boundaries, political orientation, and the interaction of each of these with experimental condition. Moreover, none of the interaction effects with potential confounding variables reached conventional levels of statistical significance. Therefore, the genomics literacy hypothesis we have proposed is the most consistent with the data we collected in this study.

If this hypothesis is correct, then one important question for future research is to explore if students construct new racial beliefs after giving up on essentialism because of genomics education. We did not produce any evidence that students replaced essentialism with some other race conception, like social constructionism, or racial colorblindness. We did not assess racial policy attitudes or affective forms of racial prejudice, either. Nor did we measure student's racial beliefs in all of the social spaces they inhabit for extended time periods. Given the limitations of our exploratory study, we cannot be sure that developing genomics literacy through genetics

education reduces all forms of racism in all of the social spaces in which it matters. Instead, these results only suggest that learning experiences emphasizing humane genomics literacy matter for reducing belief in genetic essentialism, but they matter more for students who have more SGL.

Possible Implications for 21st Century Genetics Education

If our hypothesis is correct, then genetics education could substantially reduce the prevalence of belief in genetic essentialism if it helps students to develop standard genomics literacy before helping them to develop humane genomics literacy. In the IRTC, 11% of the between student variation in belief in genetic essentialism was explained by SGL ($R^2 = .11$), 4% was explained by whether students learned from the humane genomics intervention ($R^2 = .04$), and 3% was explained by the interaction between this intervention and SGL ($R^2 = .03$). Thus, 18% of the total variance in belief in genetic essentialism was associated with genomics literacy. This means that if genetics educators harness the power of the education they provide and orient it toward the humane purpose of reducing racism, it *might* be possible to make a significant dent in the prevalence of genetic essentialism in the population of US biology students. We do not mean that education is the only factor responsible for genetic essentialism. Clearly, essentialism is a multifactorial trait that is influenced by many life experiences. Instead, we are suggesting that an education that helps students to develop *both* standard *and* more humane forms of genomics literacy could yield a small but meaningful reduction in the prevalence of genetic essentialism.

Our best prediction of the clinical significance of this reduction comes from the number needed to treat (NNT). For HISGL students, the NNT associated with our humane genomics interventions was 12, which means that we can prevent one student from believing in genetic essentialism for every 13 students treated. Thus, in a classroom of 30 students that has already developed some standard understandings of population thinking and multifactorial genetics, it

might be possible to prevent 2 students (30 ÷ 13 = 2.3) from developing agreement with genetic essentialism by engaging the class in a learning experience that develops their humane genomics literacy. We used a 45-minute computerized intervention to accomplish that objective. An entire unit that developed both of these forms of genomics literacy should be more powerful. The NNT for a full unit could therefore be much smaller, and its benefit to society much larger. The NNT might also be reduced if the K-12 genetics curriculum was organized around a learning progression that helped students to develop standard and more humane forms of genomics literacy each time they learned genetics. As a point of reference, the NNT for reducing breast cancer mortality over a fifteen year period through a combination of breast-conserving surgery and radiation treatment is 18 (Maughan et al., 2010). If future studies confirm that the NNT estimated in this study endures over longer time periods and can be replicated in other populations and in other social spaces, then genetics education may be a promising treatment for a social "cancer" like genetic essentialism.

History shows that biology knowledge makes a difference in matters of race. If the knowledge produced by biological science does not influence how people make sense of race, then why, in 1785, did Thomas Jefferson appeal to the burgeoning science of biology to justify the natural inferiority of "the Negro" as a rationalization for slavery (Smedley & Smedley, 2005)? In the 20th century, why were discredited ideas about genetics used by opponents of *Brown vs. Board of Education* in arguments to overturn it (Jackson & Depew, 2017)? Why do white supremacists continue to misconstrue genetic science to try to persuade the public that racial inequality is innate (Jackson Jr. & Depew, 2017)? Why is history also filled with geneticists who challenged racism by pointing out the flaws in genetic essentialism (Jackson Jr. & Depew, 2017)? The simple, if incomplete, answer is that genetics knowledge is regularly invoked in sociopolitical debates in order to maintain and mitigate structural inequality between races (Jackson Jr. & Depew, 2017;

Markus & Moya, 2011; Morning, 2011; Omi & Winant, 1994). If we take it to be true that genetics knowledge affects the content of these debates, then educators need to think about the kind of impact they want to have on their students' genomics literacy in the 21st century.

Notes

[1] In the supplemental, we further discuss the concept of genomics literacy.

[2] In the supplemental, we explain how the genetics curriculum can exacerbate genetic essentialism.

[3] In the supplemental, we report findings from think-alouds to suggest that active reading of the information in our humane genomics intervention differed between high and low SGL students. Consequently, fundamental genomics literacy could have mediated the interaction between standard and humane genomics literacy observed in the IRTC. For these reasons, we hypothesize that fundamental and derived genomics literacy are needed to reduce belief in genetic essentialism.

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